of extensive gastric ulcers. One sometimes finds a large ulcer of the stomach so adherent and extensive and situated so high in the body as to render gastrojejunostomy not only hazardous, but open to the serious objection that it must be made distal to the ulcer. In such cases jejunostomy is a rational procedure, and even if the ulcer be malignant in character the method furnishes a useful means of palliation.

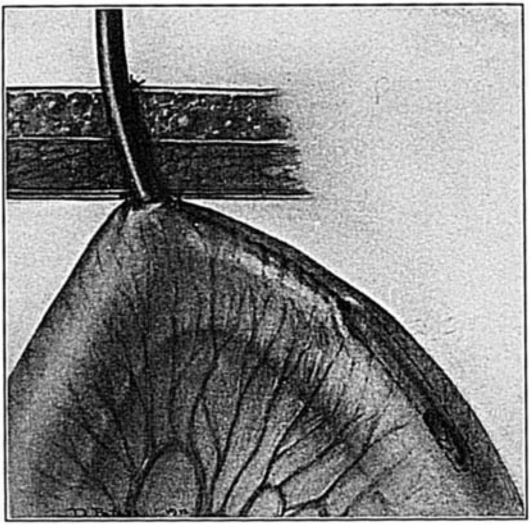

Fig. 3.—Jejunostomy: Third step, operation completed; jejunostomy fastened to abdominal wall.

Another indication for jejunostomy is in the treatment of accidental injuries to the cancerous stomach during exploration. Somewhat over a year ago while exploring a stomach which contained a large ulceration in the vicinity of the pylorus (I was trying to determine whether it was benign or malignant) I accidentally made a perforation into the foul crater of the ulcer which proved to be malignant and wholly inoperable. In attempting to repair the injury complete separation through the cancerous mass occurred. Gastrectomy was out of the question and the state of the tissue prevented the possibility of closing both ends and doing gastrojejunostomy, I therefore, reunited the cancerous tissue as best

I could with interrupted chromic catgut sutures, many of them tearing out as the tissue would not stand the strain. I then formed a sling about the line of union in the stomach with omentum, holding it in place with catgut sutures. A few rubber-tissue drains were placed about the injured stomach and jejunostomy performed. The patient was fed two months by this means. There was considerable foul discharge along the drainage tract which lasted several weeks. The catheter was removed in eight weeks, the patient gained thirty pounds in weight and lived more than a year without gastric distress and in great comfort. There was no return of obstructive symptoms in this case, although before operation the obstruction was so marked that the patient was in a chronic state of starvation and cachexia. We have observed two similar cases.

It sometimes happens, after the excision of a large ulcer of the stomach, that the organ is not in a safe condition to hold food. This is especially true of large ulcers of the posterior wall. The patients are often so debilitated that improvement in nutrition is almost a necessity in order to obtain union. Jejunostomy furnishes an easy, safe manner of giving the stomach rest during the healing process, the patient is relieved of the danger of leakage and adequate nourishment is maintained.

I am indebted to Dr. Clairmont, of Vienna, for calling my attention to the value of jejunostomy, and subsequently my interest in the operation was increased by Dr. Frank Billings, who advocated the operation on theoretic grounds, grounds which have proved correct in our clinical experience.

NOTE.—Since writing the above my attention has been called to an article in the *British Medical Journal*, January 6, 1912, by Mr. Mayo Robson in which he advocates jejunostomy in conditions of the stomach not amenable to other methods of treatment, and gives a *resume* of his cases.

PANCREATIC LYMPHANGITIS.

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"THERE is an immense amount of sepsis in medicine." These words of an eminent clinician convey an appreciation of the preponderating role played by infection in the production of pathology. The effects of infection are often obscure and far reaching, for they are indirect as well as direct, manifesting themselves at a distance as well as at the primary site, revealing their ravages at a time remote as well as immediately, and it is not too much to say that if we could attain the ambitious ideal of Pasteur of causing all the infectious diseases to disappear from the earth, that along with the so-called infectious diseases would go such a large proportion of diseased conditions, now but little considered in this connection, that our works on pathology could be condensed into the size of a primer and our occupation would be all but gone.

Still, it is our chief task to unravel the devious ways of infective disorders, since it is only by a thorough understanding of their etiology, clinical course and effects, that we may hope to palliate or cure and ultimately to prevent them. For this reason we offer this brief suggestion as to the origin of certain pancreatic swellings and inflammations.

That pancreatitis, both acute and chronic, is in practically every instance due to microörganismal invasion will hardly be denied, even though it be admitted that in certain instances systemic intoxications due to alcohol, syphilis, tuberculosis, or other unknown circulating toxins may be productive of degenerative lesions which may be classed as chronic pancreatitis. There is also reason to believe, on the basis of clinical and experimental evidence, that the retroinjection of bile into the pancreatic duct may set up acute pancreatitis of the most severe type even in the absence of microorganisms. Hlava's experiments show that the same result may follow the injection of gastric juice into the duct, though there is no evidence that this ever occurs clinically. Even in these cases infection has been demonstrated to follow quickly in the wake of the aseptic injury, and under conditions as they are found in the body, infection must practically always precede or go hand-in-hand with the entrance into the duct of any foreign material. Giving these considerations all due prominence, it still remains an incontrovertible fact that pancreatitis is for all practical purposes a consequence of infection, our conclusion being based not only upon analogy, but upon cultural examinations in acute inflammations, upon its frequent association with frankly infective processes in neighboring viscera, and upon the microscopic evidence of infection in the gland itself.

The chief point at issue is the method by which infection gains entrance to the gland. There are the following possible portals of entry: (1) The systemic circulation, through the arteries of supply. (2) By contiguity from adjacent viscera. (3) Ascending infection of the duct from the biliary tract or duodenum. (4) Through the lymphatics.

1. There is but little evidence in favor of infection by way of the circulating blood. The frequency of bacteremia and pyemia and the rarity of involvement of the pancreas in these conditions establishes our belief in almost complete immunity of the pancreas against serious infection through the blood stream. The clinical history of cases who are the subject of pancreatitis also furnishes no warrant for a belief in this mode of origin. When it does occur it must be classed as a rare exception.

2. Instances are not rare in which the pancreas is involved in inflammation by direct extension from a neighboring organ. The most frequent example is the chronic perforating ulcer of the stomach or duodenum which eventually makes its bed in the pancreas. In such cases the pancreas protects itself by granulation tissue and cellular infiltrate, and may suffer no general injury. In other instances it may show acute or chronic lesions more or less widespread. Bartels has reported a case of acute hemorrhagic pancreatitis associated with perforated duodenal ulcer, which is doubtless of this nature. Infection of the gland by direct contiguity is, however, relatively uncommon, the mechanism of which is simple, but fails to explain the great number of cases of pancreatitis which are now being recognized.

3. The great volume of discussion concerning the origin of pancreatitis deals with the noxious influences that may be brought to bear by way of the ducts of the gland. To stenosis or occlusion of the ducts, the injection of biliary or duodenal contents, and ascending duct infection have been assigned the chief and almost the sole part in the production of acute pancreatitis and the interlobular form of the chronic disease. Experimentally acute and chronic disease have been reproduced both by infection and by injection of the ducts. Clinically our attention has been centred upon the association of the disease with disease of the bile passages, and it has been natural to suppose that in the association of terminal facilities of the common bile duct and the pancreatic duct we have a ready means and explanation of infection of the pancreas, the affections of the latter being quite commonly accepted as being secondary to disease of the biliary tract in the majority of instances.

There are certain objections to this facile explanation. First, it is observed that the pancreas is often diseased without demonstrable disease of the biliary apparatus. In the senior author's experience about one-third (36 per cent.) of all instances of chronic pancreatitis, and 25 per cent. of acute cases were not accompanied by demonstrable disease of the biliary passages. Most investigators have laid stress upon the more frequent association of pancreatitis with gallstones, giving almost no attention to the minority of cases in which not only gallstones but even other evidences of biliary tract infection were absent. Truhart alone found that a smaller percentage of patients with acute pancreatitis had gallstones than those who died from other causes. Desjardins supporting the belief in ascending infection, attempts to explain this by assuming that the same infection gaining entrance into the ducts of the biliary or pancreatic system may cause at one time cholangitis, or at another, inflammation of the duct of Wirsung. This is merely an assumption unsupported by clinical evidence.

A strong objection to the similarity of causes of the two conditions is their lack of parallelism according to sex. Nothing is better established than the preponderance of cholelithiasis among females, the ratio roughly being about three to two. In the series of cases of pancreatitis above mentioned the ratio was reversed, and though percentages differ, the experience of operators the world over shows that males are much more prone to this disease than are females. This fact, while not in itself conclusive indicates that probably we have laid too great stress upon the similarity of origin and intimate relations of pancreatitis and biliary disease, and directs the attention to further inquiry into the method of origin of pancreatic affections. This additional factor and one which has received altogether less attention than it deserves, we believe to be:

4. Infection by way of the lymphatics. The validity of this explanation depends upon the existence of primary infectious processes so situated with reference to lymphatic vessels that the infection may more or less readily reach the pancreas through these channels.

Of the four great sources of intra-abdominal infection, the appendix, the Fallopian tubes, the gall-bladder, and the pyloric region, the latter two are situated in close proximity to the pancreas. If it be shown that there exists an inter-communication of the lymphatics of the pancreas with these two danger points, from our knowledge of the behaviour of the lymphatic channels as avenues of infection it would seem not unlikely that the pancreas would suffer secondary involvement. Not only the grosser lesions of the gall-bladder and juxtapyloric region, such as cholecystitis and gastric or duodenal ulcer should be considered in this connection, but also those more frequent minor infectious processes which pathologically are termed catarrhal and clinically usually fail of recognition, except under the name of indigestion or dyspepsia. In the gall-bladder we are accustomed now to recognize extremely mild grades of inflammation of the organ by the presence of ever so slight thickening, diminution in lustre, increased opacity, or inspissation and tarry character of its contents. The same scrutiny should be directed toward the stomach and duodenum where slight thickening, local puckering, or distortion and increased opacity may reveal the present or past existence of inflammation of the wall. As evidence of previous inflammation we have observed dimpling of the wall at times suggesting the starting point of an intussusception. Again, there have been delicate adhesions easily to be overlooked, and even less prominent is a peculiar streaked, opaque appearance affecting a very limited portion of the wall and often running off toward adjacent peritoneal attachments. Of course, the grosser cicatrices of healed ulcers are readily apparent, but these less conspicuous appearances should be noted as indicating, —in many instances at least, the previous existence of interstitial inflammatory processes. Catarrhal inflammation, fissures, and even early ulcers may exist without external evidence of their presence, and yet as a portal of bacterial entry their importance cannot be denied as is shown by the enlargement of the regional lymphatic glands. To those abreast of the revelations of surgery in the upper abdomen no argument is needed to convince of the great prevalence of infectious processes, the potential gates of entry of organisms into the lymphatic paths of this region. It remains to show the intimate relationship of these efferent lymphatics with those of the pancreas, and our clinical basis for believing that pancreatitis may originate in this way.

ANATOMICAL CONSIDERATIONS. The brilliant study by Bartel of the lymphatics of the pancreas must be consulted in order fully to appreciate their anatomy. This work was carried out both upon animals and human subjects, and from it we draw freely. The pancreas, unlike certain other organs, possesses no great hilus through which pass the afferent and efferent blood and lymph vessels. The lymphatics emerge at various points along its surface, and run to the regional glands, to neighboring trunks or plexuses. This arrangement should be especially noted, as it leads to a very important deduction.

It may be pointed out that by reason of its retroperitoneal situation it bears a close relation to the thoracic duct and to many trunks which empty into it from the visceral and parietal lymphatics of the abdomen. As regional glands of the pancreas, demonstrated by the Gerota method of injection, Bartel describes: The pancreaticosplenic and superior pancreatic, the superior gastric, hepatic, pancreaticoduodenal (anterior and posterior), mesenteric, mesocolic, inferior pancreatic, and peri-aortic (Figs. 1 and 2). Once he demonstrated a direct communication between a pancreatic lymphatic and the lumbar trunk, but regards this as a rarity. He never succeeded in showing a direct communication with the thoracic duct, though this had been previously described by Hoggan. To all the above groups of glands the pancreas sends lymphatic branches which may anastomose in the cellular retroperitoneal tissues with the lymphatics from the stomach, duodenum, spleen, liver, gall-bladder and bile ducts, colon, and even the left suprarenal. Probably still other intercommunications exist which were not demonstrated. Lymphatic borne infection from these areas, in order to reach the pancreas, must in most cases stem the efferent current from the pancreas, and force the valves. Here, as elsewhere in the body, the lymphatics are wonderfully efficient in preventing this outcome. Were it not true, our chief organ of digestion could not escape grave damage in a large percentage of cases, and pancreatitis would become one of the "Captains of Death."

Only when the inter-communications of the pancreatic lymphatics with those of adjacent organs are most intimate, short in their course, and unprotected by intervening lymphatic nodes does peril commonly arise. This most intimate relationship Bartel has shown to exist with the lymphatics of the adjacent duodenum, and more recently Franke has demonstrated that the same is true of the lymphatics coming from the gall-bladder

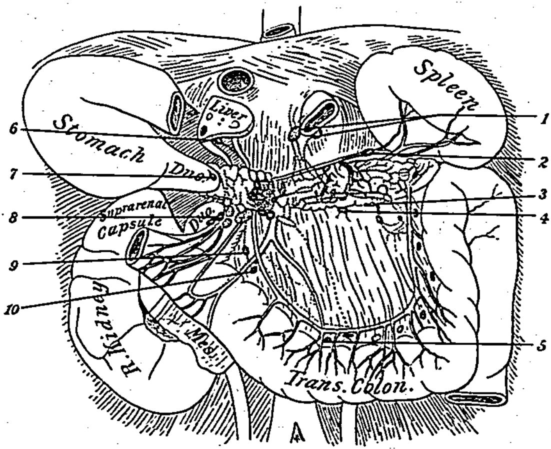

FIG. 1.—Prematurely born male child. 1, superior gastric lymph nodes; 2, superior pancreatic lymph nodes; 3 and 4, inferior pancreatic lymph nodes; 5, mesocolic lymph nodes; 6, hepatic lymph nodes; 7 and 8, pancreaticoduodenal lymph nodes; 9, mesenteric lymph nodes; 10, mesocolic lymph nodes. (After Bartel.)

How closely the finer lymphatics of the adjacent duodenum are applied to the pancreas can be appreciated by the accompanying sketch of an actual preparation (Fig. 3). The lymphatics seen upon the surface inosculate with others coming from the pancreas. In lymphatic vessels there may be a reversal of the lymph current in the effort to establish collateral circulation about points of thrombolymphangitis, and thus infection may be carried into the pancreas. An infection which is sufficiently severe would not even wait for a reversed lymphatic current, but could ascend rapidly, involving not only lymphatic structures, but the surrounding tissues as well. Such a severe infection might well cause acute hemorrhagic pancreatitis by causing necrosis of the pancreatic tissue, liberating the active ferments, and eroding the bloodvessels. Bartel was acute enough to suggest on the basis of these observations, that pancreatitis might originate in this manner, but had no clinical material to support his inference, which received scanty consideration. Arnsperger has more recently, on clinical grounds, offered a similar suggestion with reference to the association of pancreatitis and gall-bladder disease, and this view receives support from the work of Franke, who showed, also by Gerota injections, that the lymphatic vessels of the gall-bladder run to glands which lie to the left of the head of the pancreas near the choledochus, and on the right near the hepatic artery. On the way the greater part of both paths pass a gland at the neck of the gall-bladder. This gland is not, however, constant in occurrence. By injection from the gall-bladder he has filled a plexus of lymph vessels situated upon the posterior surface of the head of the pancreas. The intrinsic lymph

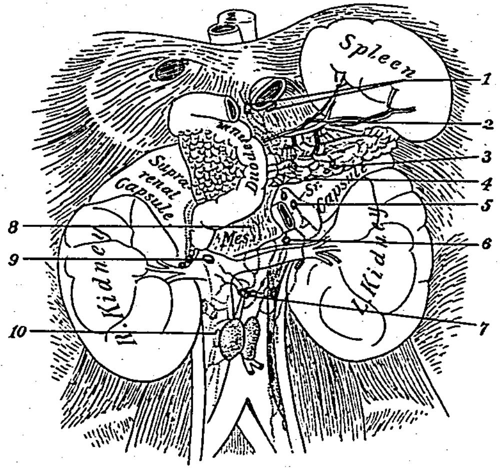

FIG. 2.—1, Superior gastric lymph nodes; 2, superior pancreatic lymph nodes; 3 and 4, inferior pancreatic lymph nodes; 5 and 6, left paraaortic lymph nodes; 7, preaortic lymph nodes; 8, from the mesenteric lymph nodes; 9 and 10, right para-aortic lymph nodes. (After Bartel.)

vessels of the pancreas run in the interlobular septa, and it is here that the effects of this type of inflammation should be most manifest. This corresponds with the fact that the form of pancreatitis usually associated with inflammatory lesions of neighboring viscera

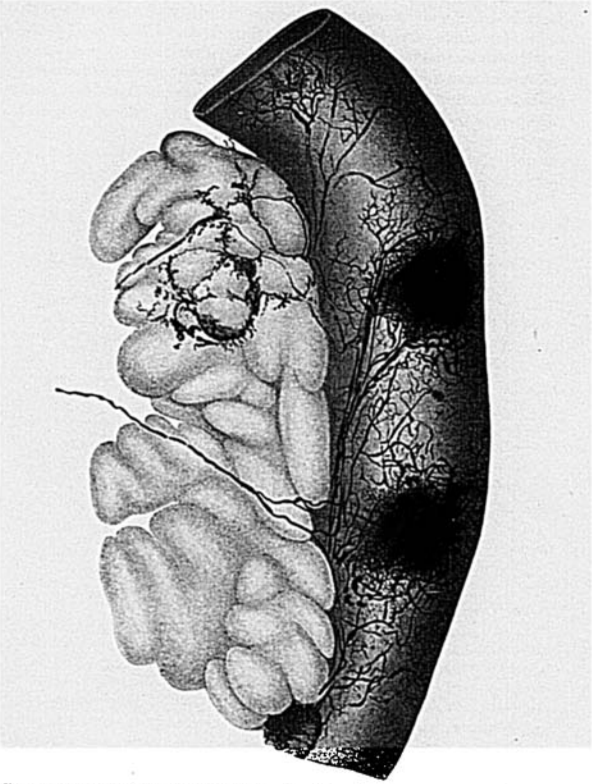

FIG. 3.—Duodenum and pancreas of newly-born dog, injected to show lymphatics. In many places an intimate connection exists between the pancreatic and duodenal lymph systems. (After Bartel.)

is the interlobular type, as contradistinguished by Opie from the interacinar sclerosis which appears to bear no such relation to local inflammation. Of itself, however, this observation does not speak for a lymphatic origin of pancreatitis since the pancreatic bloodvessels and ducts also run in the interlobular tissue, and if inflamed would produce interlobular sclerosis. A most suggestive point is the following: It is observed by all surgeons that the earlier forms of pancreatitis found at operation in connection with the gallbladder or duct disease involve only the head or perhaps only a portion of the head. In other words, *the distribution of inflammation corresponds to the lymphatic distribution*, which, as mentioned above, is irregularly segmental. It does not correspond to the duct distribution, which ramifies by dichotomous division from the main accessory ducts. It is most difficult to understand on the theory of duct-borne infection why the gland should not more often be symmetrically involved, while if the lymphatic method of transmission be granted this phenomenon is readily explainable. Again, the swellings of the head of the pancreas which are so frequently encountered by the abdominal surgeon must be different from the varieties of chronic pancreatitis described by the pathologist. Kehr, on clinical grounds, has surmised that such a distinction exists. Chronic pancreatitis, which is characterized by interlobular or interacinar deposits of fibrous tissue, is no more curable than is chronic nephritis, or cirrhosis of the liver. On the other hand, it is characteristic of the pancreatic swellings associated with biliary disease to subside with the subsidence of the biliary infection. This swelling must, therefore, be due to edema, congestion, and absorbable infiltrates. Its subsidence on cure of the primary infection would be analogous to that which occurs in the treatment of primary foci of lymphatic infection elsewhere in the body. It may be urged by those who advocate ascending duct infection that the disappearance of pancreatic involvement occurs as the result of the cessation of reinoculation with infected bile, and it is true that of itself this observation does not speak for either mode of infection. In many of these cases, however, Nature attempts by her own method of injections to point the course of events. In this form of pancreatitis operated upon at the fortunate time it is possible to demonstrate the chain of infection; infected gall-bladder, enlargement of the cystic gland, enlargement of the glands at the head of the pancreas, and swelling of the regional lymphatic distribution of the pancreatic head. Since we have been looking for these glands it is remarkable how constantly they have been found in this condition. One which appears to be especially constant in position and enlargement is situated just to the right of the choledochus, where it passes beneath the duodenum.

We have spoken of this condition chiefly in connection with the gall-bladder because it is here most easily capable of recognition, but it is our belief that lymphatic infection from the duodenum may play a considerable role in chronic pancreatitis. Not a few instances of the association of duodenal ulcer and pancreatitis are on record, while catarrhal duodenitis may also contribute a share. The greater frequency of duodenal disease in males would correspond with the sex incidence of pancreatitis, which is not true of biliary disease as previously mentioned.

It is possible that still other organs may at times furnish infection to the pancreas, but the biliary tract and the duodenum seem to be the most probable primary sources on anatomical, clinical, and pathological grounds.

To this early and remediable stage of pancreatic inflammation, in order to distinguish it from the advanced types commonly designated as chronic pancreatitis, it would be well to apply the term pancreatic lymphangitis. The reasons why pathologists have failed to recognize this condition are probably to be found in the fact that the postmortem lesions are slight and can easily be confused with that autodigestion of the organ which ensues at once after death, and often renders examination of its tissues unsatisfactory, and also because the clinician has not previously called attention to the necessity for search for the minor pathological changes. Moreover, the material has been small since it is essentially a process of living pathology. It is not of itself an immediately fatal disease. It may subside with the removal of the primary source of irritation as above pointed out, and when of long standing or frequent recurrence it leads to the deposit of interlobular fibrous tissue, in other words to chronic interlobular pancreatitis.

At the present time the diagnosis can be made only by the surgeon who can inspect and palpate the gland. The treatment is that of the primary focus of infection whether cholecystitis, cholelithiasis, ulcer, or duodenitis. In cholecystitis cholecystostomy is the operation of choice. A gall-bladder damaged to the extent that recovery and permanent cure are doubtful, should be removed. When the pancreas presents marked alterations cholecystoduodenostomy is superior in its results, though the mortality of operation should decide in favor of a simpler procedure unless there are clear indications for its performance.

For the evidence of this condition it is necessary to go to the operating table. We have not yet been fortunate enough to encounter a case complete in ante- and postmortem detail. The following case is cited to show the extent to which the disease may progress. The pathology is too far advanced and the etiology too obscure to prove its lymphatic origin in this instance. The glandular involvement, however, is similar in many respects to that observed by us in a considerable number of earlier cases where the considerations just mentioned lead to the belief in a lymphatic origin.

D. G., aged thirty-seven years. Admitted to the German Hospital, January 6, 1912. Died January 19, 1912.

Chief complaint, cramps in upper abdomen, radiating into flanks and to right side of back.

Family History. Mother died of tuberculosis, otherwise negative.

Personal History. Tea, coffee, and tobacco in excess; alcohol in moderation; denies venereal diseases. In March, 1910, he was admitted to the Pennsylvania Hospital suffering with pain beneath right costal margin radiating to the back. The pain was worse two or three hours after eating. Constipation was a marked symptom, and he had lost considerable weight. At operation cholecystitis was found and cholecystotomy performed. In July of the same year he was again operated upon for recurrence of symptoms and cholecystoduodenostomy performed. In June, 1910, he was again in the hospital with the same symptoms plus jaundice, chills, and fever. Cholelithiasis was diagnosticated, but he was not operated upon. In November, 1910, after intermittent attacks resembling those of stone in the common duct he came to the German Hospital and was again operated upon. Chronic pancreatitis, chronic cholecystitis, and a stone in the common duct were found. The stone was forced into the duodenum. The cholecystoduodenostomy opening had become obliterated; cholecystoduodenostomy performed. Recovery was uneventful, and he was improved for a few weeks, when his trouble recurred. More recently he has been having chills, fever, and jaundice.

Physical Examination. A poorly nourished man of nervous appearance. Skin moderately jaundiced. Head and chest negative.

Abdomen: Scar of old incision through upper right rectus. In this region and in the epigastrium there is marked tenderness on pressure, but no rigidity or mass.

Urine shows a very faint trace of albumin, and a few hyaline casts, otherwise negative.

Blood: Hemoglobin, 73 per cent.; erythrocytes, 4,050,000; leukocytes, 7300; polymorphonuclear neutrophiles, 73.5 per cent.; lymphocytes, 26 per cent.; eosinophiles, 0.5 per cent.; coagulation time, 8 minutes.

Stool: Dark green and practically non-odorous; fluid; neutral reaction; a trace of bile, occult blood positive to benzidine and guaiac tests. Azotorrhea and steatorrhea marked.

Cammidge reaction positive.

Operation, January 13, 1912. Dr. Deaver. Ether anesthesia. Excision of old scar in upper right rectus region; adhesions of stomach and right lobe of liver to incision; hepatic flexure of colon adherent to right lobe of liver; great omentum adherent to lesser omentum; small whitish tubercles found on small intestines; some adhesions between ascending colon and parietal peritoneum; liver covered with a few whitish nodes which are subserous;

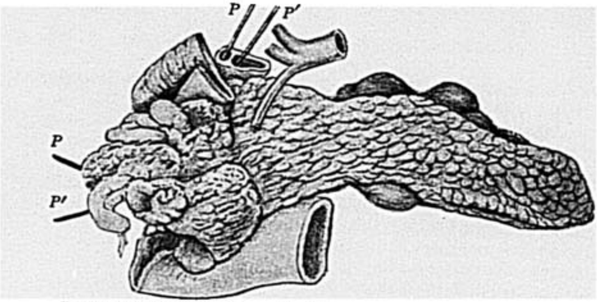

FIG. 4.—Anterior view of pancreas: *P* and *P′*, probes in choledochus and fundus of gall-bladder near gastro-enterostomy opening. Celiac axis adherent to upper margin but not in true relation. The nodules adherent to the head and upper and lower margins of the pancreas are hyperplastic lymph nodes.

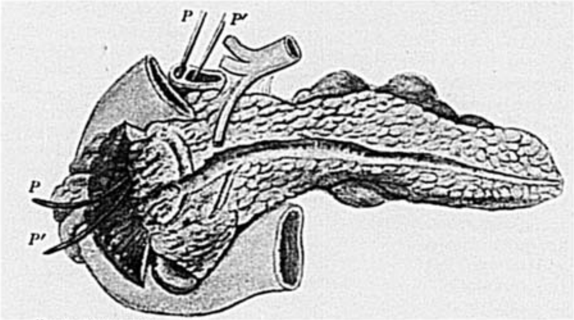

FIG. 5.—Anterior view with duct of Wirsung opened throughout its length and a window in duodenum.

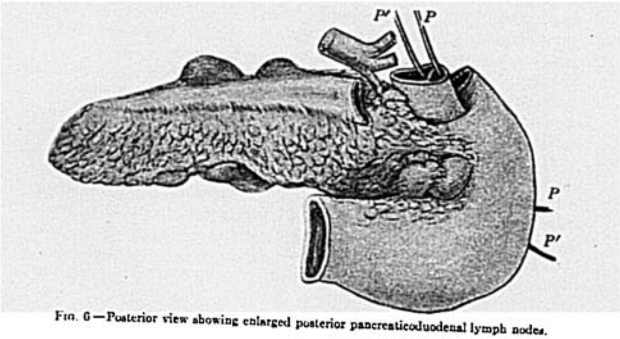

FIG. 6—Posterior view showing enlarged posterior pancreaticoduodenal lymph nodes.

old cholecystoduodenostomy still patulous; head of pancreas enlarged and hard; felt semicystic; foramen of Winslow, though occluded, was forced open; small glandular enlargement at junction of the supra- and retroduodenal portions of the common duct; head of pancreas opened by inserting scissors and withdrawing them opened (Hilton's method). Much hemorrhage followed this; was controlled by a piece of selvage gauze and one suture of iodine gut; duodenum incised and old cholecystoduodenostomy proved patent; much bile-stained material escaped from opening; head of pancreas seemed to be obstructing the duodenum posteriorly from pressure; duodenum closed with catgut baseball suture and a Lembert of linen thread; pancreas at site of incision sutured to parietal peritoneum; one cigarette drain anterior to gastrohepatic omentum; one rubber tube at foramen of Winslow; another rubber tube anterior to this and wound closed in layers.

Following the operation there was profuse brownish drainage from the wound. The jaundice disappeared. He became more emaciated and gradually weaker and died January 19, 1912.

Autopsy through the incision permitted removal of the pancreas and adjacent tissues. The head of the organ was somewhat smaller than normal, and appeared necrotic. The selvage gauze in the pancreatic wound had ulcerated into the duodenum at the site of the ampulla of Vater. A fistula existed at the site of the exploratory incision into the duodenum. A striking feature was the occurrence of many enlarged lymph nodes along the upper margin of the pancreas. A few were present along the lower border, and in relation to the head both anteriorly and posteriorly where embraced by the duodenum.

Microscopically the tail of the pancreas was the seat of a moderate grade of interlobular sclerosis. The parenchyma was mostly degenerated, probably, chiefly due to autodigestion. The islands of Langerhans were intact. The head of the organ showed marked interlobular pancreatitis and an acute suppurative process superimposed. The parenchyma was necrotic. The islands of Langerhans persisted but were involved in the acute necrotic process. A section made through a small villous patch at the junction of the gall-bladder and duodenum (at the cholecystoduodenostomy opening) showed a marked glandular proliferation which had begun to invade the deeper tissues. The process had not extended more than 0.5 cm. from its starting point, and no metastasis could be demonstrated. The lymph nodes showed chronic and acute hyperplastic lymphadenitis.

We append this history in outline with illustrations because it shows the situation and appearance of the lymph nodes in many cases of pancreatic lymphangitis. It may well be stated in connection with this case that the lymphatic involvement was secondary to the disease of the pancreas, and indeed this is true in part at least.

We have, however, observed not a few cases in which regiona. enlargement of the lymph nodes was present without involvement of the pancreas so far as could be demonstrated. Again, they have been seen in conjunction with gall bladder and duodenal disease, the pancreas still being unaffected. Finally, in still other cases, this appearance has been associated with sclerosis and nodular thickening of the pancreas which we have come to believe represent the communication of infection to the pancreas by way of the related retroperitoneal lymphatics.

CHRONIC CHOLECYSTITIS.[1]

BY J. RUSSELL VERBRYCKE, JR., M.D.,
WASHINGTON, D. C.

MACCARTY,[2] from the study of 365 cases of cholecystitis in which cholecystectomy was performed at St. Mary's Hospital, concludes that the different lesions of the gall-bladder are not definite entities, but are different degrees of the same process of reaction to irritation. He divides the cases into 8 groups: (1) Acute catarrhal; (2) chronic catarrhal; (3) papillomatous catarrhal; (4) malignant papillomatous; (5) carcinomatous catarrhal; (6) chronic cholecystitis; (7) chronic cystic; (8) purulent, necrotic. Two hundred and fifty-two, or 67 per cent., of the 365 cases were accompanied by gallstones. The chronic cases showed a large proportion of stones, while the early catarrhal conditions did not necessarily occur associated with stones.

It is to the large class of mild catarrhal cholecystitis, with or without stones, that I wish largely to confine this paper. How many people are thus affected is becoming more and more appreciated, due largely to the work of such men as Moynihan, the Mayos, Deaver, and a number of German surgeons and pathologists. By some the number of individuals having gallstones is overrated, but the great mass of physicians have been slow to grasp the real frequency of cholecystitis with or without cholelithiasis.

In Germany, as a result of the study of hospital autopsies, it has been estimated that of those dying in hospitals from all causes, 10 per cent. have gallstone and 13 per cent. cholecystitis. In England and this country it has been claimed by some that 6 per cent. have gallstone disease. Wm. J. Mayo[3] is more conservative and his conclusions are more reasonable. He thinks that 3 per cent. is probably in excess of the truth and that one-half of 1 per cent. is a fairer estimate of the frequency of gall-bladder disease at all ages, though he says that 5 to 8 per cent. of women and 2 to 4 per cent. of men have gallstones after the age of fifty. Of 1224 women operated upon for uterine fibroids at St. Mary's from 1901 to 1910, 7½ per cent. had gallstones.

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[1] Read before the Georgetown Clinical Society, November 6, 1911.

[2] Jour. Amer. Med. Assoc., liv, 2004.

[3] Ibid., lvi, 1021.

Waterhouse[4] believes that the majority of the fat, flatulent women seen at the clinics so constantly, without obtaining much relief, are really gallstone sufferers.

There is little doubt of the correctness of the theory that gallstones are produced through the action of attenuated bacteria which produce a desquamation of the epithelium of the gall-bladder, this serving as a nucleus upon which the cholesterin and other salts are deposited. Baemeister[5] holds that simple stagnation of the bile, *per se*, may cause cholesterin to crystallize out and build up one or more stones by radial formation without previous inflammatory processes being present. But, he says that after the stone is formed it may produce irritative inflammation.

Moynihan[6] has described a condition in which fine grains of calculous material are embedded in the mucosa. The wall of the gall-bladder may appear normal, being thin and pliable. Particles of the grit are also found in the bile, but no stones.

Why cholecystitis should develop in some individuals and not in others cannot be fully explained at present. Bacteria in health can be, and are, excreted through the gall-bladder. Some conditions must be present favorable to their development in the gall-bladder. How does excessive intestinal putrefaction influence the production of cholecystitis? Is it because of an increased number of bacteria carried by the portal vein to the liver in this condition? Why do gallstones and obesity so often come together? How is it that cholecystitis occurs so frequently in pregnancy and that a preëxisting stone often ceases to be quiescent at that time? These and other questions can only be answered tentatively. The influence of sedentary life and overeating is somewhat easier to explain.

A cholecystitis is probably nearly always present before stone formation, but after the appearance of the stone each condition is made worse by the other. The early symptoms are due to the inflammation whether a stone is present or not.

Gall-bladder disease is a hard malady to diagnosticate, hardest in the first stages before stones have formed and in the cases where a mild catarrhal process has continued for years without producing colic or jaundice. Such a condition may persist for two to six years or even longer. Stones tend to stay in the bladder. After colic has occurred or jaundice is present the diagnosis is easy, have come to recognize the source of some of the most destructive and sometimes fatal general septic infections, contributes an article upon general hygiene of the mouth and disorders of the teeth and gums. Too much emphasis cannot be placed upon the importance of including this subject in a work on general medicine. This article deals with those subtle diseases of the teeth and gums often overlooked by astute consultants and clinicians. The writer might perhaps have insisted upon this fact more forcibly and pointed out some of the joint diseases and other septic manifestations far removed from the real and curable cause.

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[4] Lancet, May 8, 1909.

[5] Münch. med. Woch., May 11, 1909.

[6] Annals of Surgery, December, 1909.

The treatment of nervous diseases and diseases of the mind will be turned to with much interest. This section, taken with the section on hydrotherapy and mechanotherapy which appeared in the first volume will be found to offer much practical assistance to the general practitioner. I am not competent to speak for the alienist and neurologist.

Lack of space prevents further detailed consideration. The general practitioner, and in many instances the specialist, will find in these three volumes a reliable guide, and future authors of systems on treatment will do well to employ this as a model. The publishers have made attractive volumes, which in spite of their size and number of pages, are surprisingly light, and lie open well at almost any portion of the book. C. N. B. C.

APPLIED ANATOMY. By GWILYM G. DAVIS, Associate Professor of Applied Anatomy, University of Pennsylvania. With 630 illustrations, mostly from original dissections and many in color by Erwin F. Faber. Philadelphia and London: J. B. Lippincott Co., 1911.

THE object of this work is to show the relation of structure to function, whether it is normal function or function disturbed or impaired by injury or disease. The anatomy of the human body is studied in order that medical and surgical treatment may be intelligently applied, and consequently a book of this character possesses greater value than a mere treatise on anatomy, unless the latter is regarded simply as a storehouse of facts. There is a good deal of exaggeration of the value of a minute knowledge of anatomy to the surgeon, but there is no gainsaying the fact that in certain operations, such as tendon and nerve transplantation, plastic operations on joints, dissections of the neck for tuberculous nodes, hernia operations, and some others, the operator will do better work if his anatomical knowledge is exact.

This book seems to accomplish the task admirably, and includes a variety of anatomical and surgical facts, balanced and associated so as to make an intelligent, interesting, and stimulating presentation of applied anatomy. There are, of course, omissions and statements to which others might take exception, but such must be unavoidable in the preparation of a work of this size by one writer. The purely anatomical portions seem more satisfactory to the surgeon than do the surgical portions, perhaps to the anatomist the reverse might be true, as it is disappointing, when referring to the applied anatomy of a certain operation or disease, to find the technical steps in the operation discussed briefly and without direct connection to the anatomy of the region. For instance, the extension in cancer of the lip is insufficiently treated, and in excision of the tongue no description is given of the anatomical points involved in removal through the neck; the description of excision of the rectum includes removal by the perineal or sacral routes, but no mention is made of the anatomical points involved in removal through the abdomen or by the so-called combined route, a matter of extreme importance. In the description of hernia the antiquated divisions of congenital inguinal hernia are well discussed, as are the various anatomical layers and structures entering into the formation of the canal and rings, but the relation of the sac to the cord, of the femoral vein to Poupart's ligament, etc., are not clearly given. The author seems at his best in the description of the joints, the chapters upon the shoulder and hip being especially praiseworthy; the abdomen, however, is not nearly so satisfactory.

The illustrations are clear, beautiful in execution, and have the faculty of showing what they are intended to show in the great majority of instances. A few, such as the one showing excision of the thyroid gland (Figure 184) convey no idea which might be useful. The general appearance of the book, its binding and printing, is excellent. G. P. M.

THE MORTALITY OF ALCOHOL. By EDWARD BUNNELL PHELPS, M.A., F.S.S. Pp. 75. New York: Thrift Publishing Co., 1911.

THIS is a statistical study of the causes of death in the United States by alcohol. The author is well qualified for such work, inasmuch as for over twenty years he has edited one of the leading insurance magazines in this country, and in other ways has maintained his interest in the cause of death. He gives comparative tables in which alcohol may have been the cause of death, either as a primary or secondary factor and compares his results with those of the United Kingdom. He comes to the conclusion that the mortality in which alcohol figures directly, indirectly, or even remotely is about 5 per cent., or about 6 in 100 deaths. This is somewhat lower than the estimate given by others, but, taking into consideration the difficulties which enter into such calculation, it is probably a just estimate. The work is well done, and is an interesting contribution. T. H. W.